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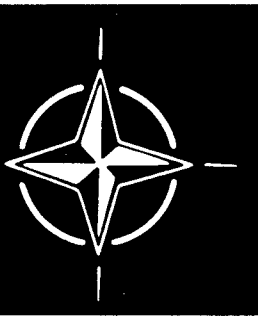

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NEUROPATHOLOGY AND CAUSE OF DEATH IN U.S. NAVAL AIRCRAFT ACCIDENTS *

by

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SUMMARY

A frequent cause of death in naval aviators is hypothesized as drowning, associated with "acceleration concussion" perhaps due to neck stretch. Present autopsy procedures on aviation accident fatalities could be improved in order to investigate this hypothesis.

Aircraft accident fatality data for the U. S. Navy are presented as a measure of the population at risk and recent data from the literature which might explain the causative mechanism of acceleration concussion is presented. Recommendations for improved standard autopsy protocols for aircraft accident fatalities are presented.

INTRODUCTION

Crash fatalities to aviators represent a continuing drain upon available resources which to some considerable extent is preventable. Intensive study of accidents and injuries, both fatal and non-fatal, has been carried out by the U. S. Navy in aircraft accident boards for many years with the results of the investigations coded and stored in computers by the Naval Safety Center. This represents a rich resource for determination of the epidemiology of fatal injuries. The number of accidents of unknown cause amounts to 40% considering only fatal accidents, as Mason mentioned in 1962 (1). The same author continues: "In these circumstances, facts can be established by deductive reasoning only and it is reasonable to suppose that an experienced appraisal of the findings within the dead body will give evidential support comparable to that based on the findings in the 'dead' machine".

EPIDEMIOLOGY

In 1963, Ewing observed that naval aircraft crashes might be associated with concussion due to neck stretch (2). This was suggested by the work of Friede (3, 4, 5). In the discussion after the paper, CAPT Richard Luehrs MC USN stated that: "In recent years, I have seen 11 planes float by the side of the carrier with the pilot sitting in the cockpit making no attempt to get out. In this type of accident, there is no place where the pilot can hit his head unless it is laterally. I think it would be very unusual that a flier would hit his head laterally when the plane is moving straight ahead. I do not think he is hitting his head on anything. I think he is breaking his neck with the weight of the helmet when the moving plane hits the water" (2) - p 118.

This represented the experience of just one flight surgeon albeit a most experienced one. Other similar cases may not have been reported by flight surgeons.

Such cases would not ordinarily be reported or coded by the Safety Center as such, since such a pilot would be recorded only as lost at sea, with only a rare recovery of the remains (about 90% are not recovered) (6).

In order to further investigate this problem, Naval Safety Center was queried as to various statistics. Their reply (reference 6) was analyzed by Ewing.

These data showed that in the five fiscal years, 1959 through 1963, there were 6,974 individuals involved in aircraft accidents involving ejection, bailout, collision with ground and collision with water (which also includes ditchings), of whom 89% were exposed to crash impact accelerations of collisions with ground or water while only 11% avoided them by ejection or bailout. Furthermore, 70% of all fatal jet embarked accidents, and 88% of all fatal prop embarked accidents were collisions with water, while only 22% of all jet embarked accidents and only 51% of all prop embarked accidents were collisions with water. The total number of fatalities during this period from collision with water alone was 226.

With this evidence of the severity of the problem in the Navy, a working paper was prepared in which it was attempted to determine the cause for the high fatality rate (7). In part, it stated:

"In attempting to determine a reason for the experience quoted in the first paragraph, a study of the literature combined with a study of the aviator's situation during a crash provided the following:

1. The aviator's head and neck are unrestrained, permitting relative motion in a deceleration event between the restrained torso, and the unrestrained head and neck.

2. The crash helmet, worn in all carrier aircraft, has a center of gravity location which shifts the center of gravity of the head-helmet mass superiorly and anteriorly. This would tend to increase the rotational moments of the head on the neck.

3. The crash helmet adds 50% in weight to the normal weight of the head, thus increasing markedly the force exerted on the neck in a deceleration event.

4. According to reference (3), 'both the typical symptomatology and the pathology of acceleration concussion can be reproduced by other means than by applying a blow to the head; that is, all the typical signs of experimental concussion can be reproduced without applying a blow to the head; for example by cervical stretch. Cervical stretch has been shown to exist in acceleration concussion. An experimental analysis of the various mechanical factors involved reveals that stretch and flexion of the cranio-cervical junction are most important for the mechanics of concussion'. Acceleration concussion is defined for purposes of this discussion as concussion occurring in an individual with a freely moveable head (i.e., unrestrained and not resting against anything) who does not receive a blow to the head and who does not suffer cortical injury.

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* Opinions or conclusions contained in this report are those of the authors and do not necessarily reflect the view or the endorsement of the U. S. Navy nor the sponsoring organizations.

5. The neuropathology involved is a circumscribed fiber lesion at the ventral circumference of the 1st cervical segment (in cats). To demonstrate this lesion, the brain and spinal cord must be removed without transection and the region between the caudal end of the fourth ventricle and the second cervical segment should be cut serially in longitudinal sections. The intensity of fiber damage in the cord is paralleled by the extent of cell changes in the medulla oblongata, the latter arising from the former.

6. In order to study the neuropathology of concussion in any animal (including man), it is necessary to (a) cause the injury, (b) wait several days to allow axonal and neuronal changes to occur, then (c) sacrifice the animal, (d) remove the brain and cord intact, and (e) prepare tissue sections for microscopic study. Since we rarely, if ever, have this exact chronology in human aviation fatalities, we must accept the fact that the basic data on which decision must be made will be that from animal experiments.

7. In reference (2), concussion produced by sudden deceleration from a velocity of the head of 25 mph produced reflex abrogation for durations of 60 seconds or longer, and the duration of loss of consciousness always exceeded the duration of reflex abrogation. It should be noted that studies on jet aircraft have shown that the aircraft usually sinks within 60 seconds" (8).

NOTE: Rawlins, et al found that jet aircraft float only for a maximum of 1 minute, and after sinking descend to the bottom at a minimum rate of 400 ft/min thus giving a maximum of 2 minutes for the aviator to escape before being crushed by water pressure (3).

"Therefore, it appears probably that the explanation for CAPT Luehrs observations is that: (a) aviators can be rendered unconscious by acceleration concussion, probably not due to nor accompanied by cervical vertebral fracture or to head injury; (b) that such acceleration concussion can easily be produced in collisions with ground or water; (c) that the expected duration of such unconsciousness would exceed the time necessary for the aircraft to sink; (d) that the concussion produced was made worse by present helmet design and lack of head-helmet restraint; (e) that the aviators, therefore, died from drowning rather than head or neck injury per se; (f) that a majority of naval aviators suffering collisions with ground or water are exposed to the risk of this hazard; (g) that a majority of fatalities from jet and prop embarked accidents occur in accidents (collisions with water) where this risk is present; (h) that only 10% of the remains of such fatalities are recovered so that no pathological study of the remains is possible; and (i) that therefore this problem has previously been unrecognized in naval aviation as such.

In attempting to find a means of preventing acceleration concussion, reference (9) states that -- 'these high linear accelerations did not result invariably in concussion when the cervical spine was immobilized. Acceleration of 930G failed to cause concussion. It became evident that concussion was produced more easily when the head was free to move on the neck'.

Therefore, research and development effort must be initiated on a high priority basis with the following phases and objectives:

1. Determination of exact information, usable by engineers, concerning the production of acceleration concussion in animals.

2. Utilization of this information to design, manufacture, and evaluate protective equipment (such as head-helmet restraint gear) which will protect against this hazard in appropriate aircraft systems.

3. Evaluate the equipment answers to the problem on human subjects on (experimental) acceleration devices.

---- This problem, though not peculiar to the Navy, is of greater importance to the Navy than to most other agencies (due to the high proportion of fatalities in embarked collisions with water). A multidisciplinary approach is mandatory, including such diverse fields as neurosurgery, mechanical engineering, research aviation medicine, human engineering, neuropathology and impact physiology.

Although it is well known that a certain number of Naval aviation accidents are of such a nature that the pilot has no chance for survival, it is possible that a very large number are survivable that are not now being survived. It is quite probable that a satisfactory solution to the problem delineated in this paper can decrease the fatality rate in Naval aviation more than any other single improvement in sight, with the possible exception of a successful rocket ejection seat and even that would be limited to jet aircraft."

This early, somewhat simplistic analysis, contained several concepts leading to the work which followed.

In order to get more precise data of more recent data, a study was undertaken of FY 1967 to determine all aviator losses; the type of accident; cause of death; and autopsy results (if performed) using Naval Safety Center data (10). The object of the study was to investigate the possible means which might have prevented the fatality, given the knowledge of what caused it. Many results have flowed from this study, including the development of a major aeromedical research program designed to develop the human data necessary to evaluate candidate protective devices against the known causes of fatal injury. Several findings of that study are now being more thoroughly investigated, and one of these is the problem of drowning in an accident which otherwise might not have resulted in significant injury, as outlined above.

The study showed that in FY 1967, two hundred and twenty-eight (228) Designated Naval Aviators were killed, lost at sea, missing in action or fate was unknown as a result of aircraft accidents, both combat and non-combat. Of the total, two hundred and twenty (220) were either pilot or co-pilot and were thus in a predetermined location at time of aircraft accident. Of this number, only forty-one (41) ejected and two (2) bailed out. The remaining one hundred and seventy-seven (177) individuals crashed in their seats.

Ninety-six DNA's of whom 4 ejected were involved in the single accident type of collision with ground or water. Examination of known causes of death for these individuals revealed that 26 were lost with the aircraft, details unknown, 5 more were lost at sea not in the aircraft, and 4 more were drowned. Therefore, thirty-eight percent (38%) of the ninety-two (92) personnel were lost at sea, drowned or lost with the aircraft.

It is probable that some of these pilot losses were due to temporary unconsciousness or concussion due to the crash, rendering them unable to escape from the sinking/burning aircraft.

During the course of the FY 1967 study, (conducted in FY 1968), it was determined that autopsies were being conducted by general pathologists at hospitals nearest the accident site; that the examination did not necessarily include x-rays or photography prior to the autopsy, that frequently CNS examinations were not done at all; and that aviators who survived for a number of days following the crash but subsequently expired were infrequently subjected to autopsy and almost never to a CNS postmortem examination. Furthermore, remains recovered from the water after days of immersion were frequently treated in the same fashion.

Death from burning is another frequent and important hazard of aviation accidents. Thermal burns rank with craniocervical injury as the most frequent cause of aviation deaths after multiple injuries. Combined mechanical and thermal trauma frequently occur. Unfortunately, a detailed and comprehensive study of damage to the central nervous system due to thermal burn does not exist.

Some of the described pathomorphological alterations due to burns, however, are similar or identical to secondary traumatic or hypoxic alterations due to mechanical trauma.

In summary, most aviation autopsies have not resulted in satisfactory neuropathological examination of the central nervous system.

An autopsy of the crash victim must show not only the cause of death, but also a concise description of the distribution and quality of the tissue alterations in brain and spinal cord which can be considered the morphological endstates of mechanical trauma inputs including thermal injury.

In view of this analysis of the magnitude of the problem; and of the shortage of neuropathologists in the Navy; and of the lack of knowledge of aviation pathology on the part of, or guidance to the hospital pathologist, it became apparent that the most important source of human pathological material required to establish the presence or absence of the acceleration concussion histopathology, or even the possible mechanism of production of it, mentioned above, was being neglected.

It was, therefore, decided to undertake a more profound search of the literature of possible mechanisms of production of acceleration concussion to aid in making recommendations as to specific autopsy protocols of aircraft accident victims which might be more productive of evidence which could support or reject these possible mechanisms.

The purpose of this paper is to present the results of this research, along with some preliminary results of experimental studies on animals at this facility, and resulting neuropathological considerations and recommendations for improvements to autopsy protocols.

NEUROPATHOLOGICAL CONSIDERATIONS

Association of Cerebral and Cervical Injury

More attention has been drawn to the common association of cerebral and cervical cord injury. Too often the latter is overlooked. It is useful, therefore, to quote and analyse the papers which were published on this matter.

Leichsensing (1964) found that in 20 unselected autopsies of lethal head injuries, hemorrhages of the spine and spinal cord could be observed macroscopically and microscopically in every case, regardless of the severity of the head injuries or of the existence of skull fractures (11). In six cases, ruptures of the anterior or posterior longitudinal ligaments were seen. Traumatic disc ruptures occurred at the level of C4/5 and C5/6. In the majority of cases peridural hemorrhages were observed, which extended into the intervertebral foramina bilaterally, without predominence of any single segment. Only in the most severe injuries with complicated fractures of the occipital bones were ruptures of the ligaments of the atlanto-occipital joint and atlanto-axial joint seen. In cases of less severe head injuries, the caudal segments of the cervical region were more frequently involved. Small hemorrhages could be seen in the spinal cord too, in cases of minor injuries.

Gosch et al (1970) reported that petechial hemorrhages at the cervico-medullary junction were noted in football players who sustained direct "head-on" or vertex impacts when they struck an opponent (12). Similar head and cervical spinal cord injuries were produced in experimental animals on an impact track simulating this mechanism. Severe cervical spinal cord destruction could be obtained in the absence of cervical flexion and extension. Cord movement was enhanced by sectioning the dentate ligaments, which prevented these lesions. These authors postulated that the transmission of shear strains along the axis of acceleration was responsible for the hemorrhages when the elastic deformation limit of the cervical spinal cord was exceeded.

Davis et al (1971) reported a series of 50 fatal cases of craniospinal injury and reviewed them with particular attention to the pathological findings at the craniospinal junction (13). There was marked tendency for the spinal cord to be damaged in the upper cervical segments, whereas disc injuries predominated in lower cervical segments. The vertebral arteries were rarely involved. The lateral ligamentous structures were often damaged, while the transverse ligaments of the odontoid were spared. Rupture of muscles was rare, although hemorrhage into muscles was common.

A series of 146 victims of fatal traffic accidents were subjected by Alker et al (1975) to postmortem radiographic examination prior to medico-legal autopsy (14). A total of 42% were found to have radiographically demonstrable head injuries ranging from relatively simple linear skull fractures to massive skull damage. Free intracranial or intravascular air was demonstrated in more than 60%. A total of 21% had demonstrable neck injuries, most of which were localized to a single level at the craniocervical junction or in the area of the upper two cervical vertebrae. Flexion and extension studies of this area were of major importance in demonstrating the injury and locating potentially occult lesions for the forensic pathologist.

Alterations in the spinal cord near the occipito-cervical junction were reported in animal experiments in which different species of animals had been subjected to impact acceleration.

Denny-Brown and Russell (1941) and Denny-Brown (1961) have noted that occasionally animals concussed experimentally showed petechial hemorrhages in the cervical cord, and in their experiments movements of the head were purposely restricted to 4 cm after the blow to minimize this possibility of cervical damage (15, 16).

Unterharnscheidt in 1963, 1970, and Unterharnscheidt and Sellier (1966), reported on a large series of rabbits and cats who had been subjected to subconcussive and concussive acceleration both in the +Gz and +Gx vectors (17, 18, 19). They found that occasional smaller petechial hemorrhages in the gray and white substance of the spinal cord at the occipital-cervical junction could be observed. This author explained these pathomorphological findings as the result of marked stretching movements at this junction due to the impact acceleration of the skull.

Friede (1960) investigated the central nervous system of cats in which an abrupt stretch of the cervical vertebral column had been performed (3, 4, 5). The same type of brain stem damage was found as had been described following experimental concussion: a chromatolysis appeared in the large neurons of the reticular formation and Deiter's nucleus: In severe injuries it also appeared in the red nucleus and some other nuclei. The motor nuclei of the cranial nerves were not involved. He found also a significant fiber damage in serial studies of the cervical spinal cord. Thick fibers in the medioventral portions of the cervical spinal cord were affected exclusively. This damage was maximal at the atlas level

but sparse above this level. The locally defined fiber damage corresponded to the distribution of chromatolytic neurons sending their fibers through the damaged area and both findings were quantitatively related. Changes in the fiber structure were found also in cats killed by experiments.

X-ray investigations revealed an intimate spinal cord-bone relationship at the level of the damaged area of the spinal cord. In particular a straining of the spinal cord around the odontoid process occurred in forced position changes of the head and could be enhanced by a subluxation of the odontoid process.

Friede found also identical neurohistopathology in dropped cats subjected to an abrupt stretch of the cervical spinal cord and in control cats receiving a blow to the head. The chromatolysis in the neurons in both the stretching experiment and in cats receiving a blow to the head, is most probably a retrograde degeneration resulting from the fiber damage in the spinal cord. Friede concluded: "Therefore, a specific mechanism of cord injury at the atlas levels seems responsible for many instances of so-called 'brain' concussion".

Unterharnscheidt and Higgins (1969) carried out carefully controlled studies of non-deforming angular acceleration in the +Gx vector over 45° angle (20, 21). Location and quality of brain lesions was reasonably predictable from the mechanical input to the CNS. These pathologic changes consisted of subdural and subarachnoid hemorrhages; tearing and avulsion of veins and arteries in superficial cortical layers leading to rhectic hemorrhages; partial and total traumatic necroses; and rhectic hemorrhages in cranial nerves. With the exception of a few animals, the spinal cord showed small rhectic hemorrhages in various segments through its entire length, extending to the cauda equina. It was demonstrated that a difference in quality existed between primary traumatic cortical hemorrhages associated with angular acceleration and the so-called cortical contusions found in translational trauma. A difference was noted in the patterns of distribution of the primary traumatic cortical contusions encountered in angular as opposed to translational acceleration. It was demonstrated that a non-deforming rotational acceleration of the head could produce leasions not only in the brain but through the entire length of the spinal cord.

There exists no doubt for us that the alterations described by Denny-Brown and Russell (1941), Friede (1960), Denny-Brown (1961), Unterharnscheidt (1963) and Unterharnscheidt and Higgins (1969) are intravital; they are not artifacts (15), (5), (16), (17), (20, (21). The latter two authors were able to describe mesodermal-glial reactions in animals which survived for several days. In accordance with Spielmeyer, this reaction is the result of destruction of nervous parenchyma; the destroyed tissue is replaced by a mesodermal-glial scar (22). A further indication for the fact that these alterations are intravital can be drawn from the fact that the tissue damage is dependent upon the intensity of the applied forces. These tissue alterations, especially those in the lower medulla and the cervical spinal cord, are the morphological endstates of different types and intensities of mechanical trauma. But further experimental work is necessary to clarify whether these tissue alterations are a substrate which is typical and pathognomonic of cerebral concussion, or whether they are the result of different types of mechanisms which lead to stretching or extreme rotation of the occipito-cervical junction. Unterharnscheidt (1963), (1970) and Unterharnscheidt and Higgins (1960) are of the opinion that the described lesions are not a substrate which is present in an uncomplicated cerebral concussion or a commontio cerebri, but that they occur in certain injury types, mechanisms, vectors, or intensities of impact acceleration which produce extreme stretching and rotation at this junction (17), (18),(20), (21). Further evidence for this opinion can be found in new data from recent unpublished animal experiments at NAMRL Detachment, New Orleans, Louisiana, with whole-body -Gx acceleration exposures of Rhesus monkeys with restrained torso but unrestrained head and neck (23). Each of the injury types and vectors of the effective acceleration lead to a typical predictable injury pattern with respect to distribution and quality of the tissue damage.

The preponderance of evidence presented, therefore, demands that in clinical circumstances in which humans are subjected to severe acceleration of the unrestrained head, the spinal cord as well as the brain should be examined histopathologically and that the upper cervical cord should be examined en bloc with the brain.

Present Autopsy Procedures

Present instructions for Naval autopsies are not mandatory and are in effect only routing instructions for the results (24),(25), (26), (27). X-rays are not mandated for any autopsy case but only for all ejections, ditchings, or crash landings where "significant forces were present" (25).

A suggested technique for removal and examination of the spinal cord and vertebral column is presented in (27) pages 182 - 184, dating back to 1962. It was recommended that the spinal cord is best exposed through a posterior midline incision, extending from the base of the head to the sacrum. The dura mater should be left with the spinal cord. It was stressed, however, that a laminectomy should not be done on the first cervical vertebra, but that this region should be tunneled, since any interference with it will destroy the rigidity of the connection between the head and the trunk.

It was not mentioned at which level the spinal cord should be severed from the lower medulla of the brain. This is a very important point, since the area between lower medulla and upper cervical cord is in the zone of the most extensive stress, as is shown above and as demonstrated also by Clarke et al (1972) (28).

Recommendations for Standardization of Autopsy Procedures in Aircraft Accident Victims, Derived from the Studies Reported Above

The procedure enumerated below will be performed on the following decedents:

1. All personnel suffering fatal injury due to aircraft accident, including drowning victims.
2. All personnel injured in aircraft accidents who die subsequently in a hospital.
3. All personnel who die within 30 days following their involvement in an aircraft accident.

Radiographic Examination - A postmortem radiographic examination should be carried out routinely on all fatalities resulting from aircraft accidents to include (1) anteroposterior, lateral and basilar views of the skull, (2) anteroposterior and lateral views of the cervical spine, (3) tomographic studies when necessary, (4) chest, abdomen, pelvis, extremities, and other regions of the spine when considered necessary on the basis of visible injuries.

CNS Autopsy Technique

Techniques for removal of brain and spinal cord in necropsies vary to some degree. But in order to describe, evaluate, quantify and compare the morphological endstates a standardization of the techniaues used is a necessity.

As a result of the studies reported above, a more comprehensive autopsy must be performed on the remains. This should include, but not be limited to, a detailed gross and microscopic examination of all injured organs exclusive of the central nervous system,which will be handled separately as described below. It should also include a description of the status of both common and internal carotid arteries and both vertebral arteries. In case of an evident injury to these vessels, the entire specimen should be forwarded.

The brain and spinal cord down to the cauda equina must be removed in toto, leaving the unopened spinal dura mater on the specimen, using a posterior incision and laminectomy. This is quite important because the level of the cranio-cervical junction is suspected as a frequent site of fatal injury in naval aircraft accidents but ordinary autopsy procedures destroy this vital area. A cut may be made at the cervical-thoracic junction.

Section of or dissection of the unfixed fresh brain should not be performed at the autopsy table.

The specimen should then be fixed in 10% neutral buffered formalin for about two weeks and then shipped.

The pituitary should be removed and fixed in 10% neutral buffered formalin.

After photographing and describing brain and spinal cord a Spielmeyer assortment of tissue blocks for histologic examination should be cut.

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